CASE REPORT

Treat the patient not the blood test: the implications of an increase in cardiac troponin after prolonged endurance exercise

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Collapse after prolonged endurance exercise is common and usually benign. This case study reports a triathlete who suffered a vaso-vagal associated collapsed after exercise. Misdiagnosis of myocardial injury in the presence of elevated cardiac troponins and ECG anomalies led to inappropriate management and highlights the difficulty in treating the collapsed athlete following arduous exercise.

In April 2006 a 43-year-old Caucasian male club level triathlete presented to the CRY Centre for Sports Cardiology for a follow-up consultation. In March 2006, after completion of an Ironman triathlon (4.2 km swim, 180 km bike, 42 km run) in a personal best time, the athlete received a massage during which he experienced presyncopal symptoms. He received fluid resuscitation (500 ml, 1% saline) and recovered well. After a long period of standing in the heat the athlete reported presyncopal symptoms and collapsed, without syncope. Blood pressure was measured at 75/40 mm Hg, and after further fluid resuscitation (500 ml, 1% saline) he was referred to the cardiology department of the local hospital. The athlete was conscious and alert with normal core temperature, blood glucose and plasma sodium, and was otherwise asymptomatic.

On admission his ECG demonstrated minor ST-segment elevation in anterior lateral and inferior leads (fig 1) and his cardiac troponin-I (cTnI) was mildly raised at 0.06 U/l. An ECG was normal with mild, concentric left ventricular hypertrophy. The athlete underwent diagnostic cardiac catheterisation, which demonstrated normal coronary arteries. Owing to the mildly raised cTnI and minor ST-segment elevation, he was diagnosed with mild myopericarditis and advised to avoid exercise for 6 weeks.

On follow-up, 2 weeks after being discharged from hospital, the resting 12-lead ECG demonstrated similar findings of marginal ST-segment elevation in anterior lateral and inferior leads (fig 2). Echocardiography demonstrated mild concentric left ventricular hypertrophy (12 mm) in the presence of normal diastolic and systolic function. There was no evidence of myocarditis or pericarditis and the athlete was immediately cleared for training and competition.

DISCUSSION

Exercise-associated collapse is common after prolonged endurance exercise[1] and is usually benign, however in some circumstances can be severe and life threatening. Benign causes of collapse include exhaustion, postural hypotension, dehydration and leg cramps. Serious causes include hyponatraemia, heatstroke, hypoglycaemia, hypothermia, cardiac arrest and other medical conditions.[2] In the presence of a conscious, alert athlete presenting with normal blood glucose and plasma sodium, and normal core temperature, as observed in the athlete in the present case study, the likely cause of collapse is a vaso-vagal response leading to postural hypotension that may be exacerbated by dehydration.

Although the health benefits of regular, moderate-intensity physical activity are well recognised, there is a growing body of

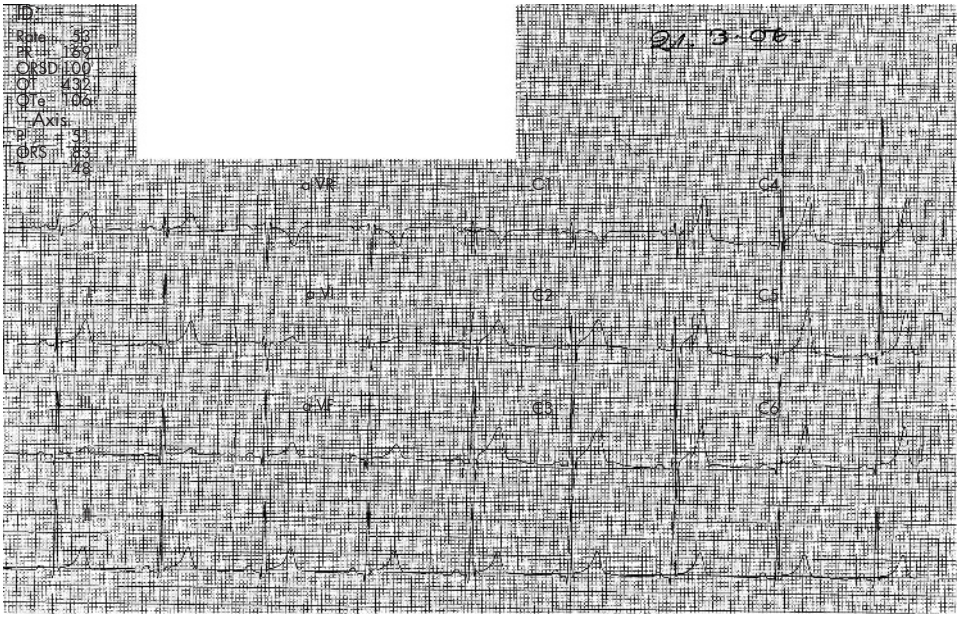

Figure 1 Resting ECG on admission to hospital.

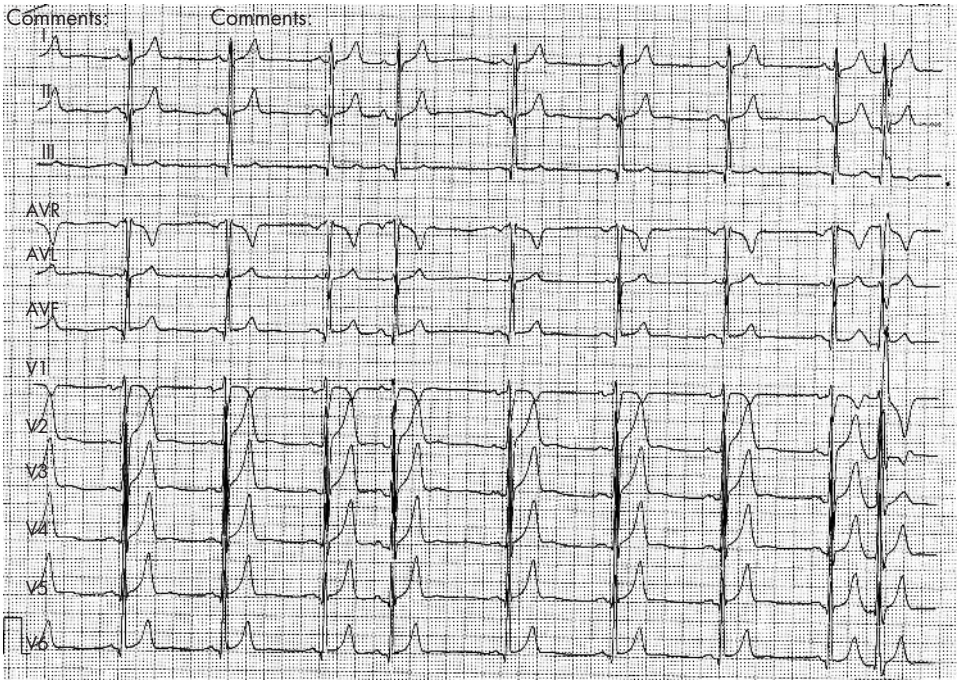

Figure 2 Resting ECG on presentation at follow-up 2 weeks after admission to hospital.

evidence demonstrating an acute reduction in cardiac function and the presence of humoral markers of cardiac myocyte damage (cTnT and cTnI) that may be above acute myocardial infarction cut-off levels after ultra-endurance exercise.[3] Among this work are a number of studies examining the Ironman triathlon.[4 5] The prevalence of increases in cTnT or cTnI, or both, after exercise is not fully known. However, in a recent study, 78% of runners investigated after a marathon presented evidence of minor cardiomyocyte damage.[6] Previous studies have suggested that these changes are likely to be physiological owing to their rapid (<24 h) return to baseline values and of no clinical significance. Indeed, the latest work from our own laboratory demonstrates a peak in cTnT at 3 h after exercise with a subsequent fall to baseline levels at 24 h after exercise (unpublished findings). These findings are in direct contrast to the kinetics of cardiac troponin release observed after myocardial infarction, where an initial release of unbound troponin is followed by a continued release of the structurally bound troponin as it degrades, resulting in a sustained rise in circulating troponin for many days after infarction.[7]

The level of cTnI seen in the athlete in the present study is similar to that previously described in athletes after ultra-endurance exercise and probably represented a physiological response to prolonged arduous exercise. The use of single measures of cardiac troponins should be viewed with caution after prolonged exercise. Serial measures would help to eliminate myocardial infarction as a cause of the raised cardiac troponins.

A number of ECG changes are seen in athletes that are associated with physiological anomalies and of no clinical

What is already known on this topic

- An altered cardiac function and blood markers of cardiac damage after prolonged arduous exercise have been extensively reported since the late 1990s.
- It is postulated that the cardiac troponins present after prolonged exercise and their rapid return to baseline (<24 h), although pathognemonic of cardiac damage, are physiological and of no long-term significance.

What this study adds

- This study highlights the difficulties faced by medical support teams and inhospital medical staff when dealing with athletes after prolonged arduous exercise.
- Despite the extensive reports of raised cardiac troponins after prolonged exercise, this case study demonstrates the lack of awareness of this phenomenon and the often inappropriate care provided.
- This study offers advice, guidance and supporting literature in dealing with this problem.

Take home message

- Care is warranted in the interpretation of raised cardiac troponins after ultra-endurance exercise.
- Serial measures of cTnT/cTnI after exercise may assist in the differentiation between underlying physiological and pathological mechanisms.
- Use of ECG screening before competition may help in the management of an athlete after exercise.
- Misdiagnosis of myocardial injury after ultra-endurance exercise and subsequent mismanagement, including admission to hospital and invasive intervention, can be unnecessarily expensive and psychologically damaging to the athlete.
- Diagnosis of myocardial injury after prolonged exercise should be made based upon all available information and not on blood tests alone.

significance.[8] The diffuse minor ST-segment elevations seen in the athlete's ECG in this study represent a normal variant as shown by their presence at follow-up examination. Furthermore, the absence of echocardiographic evidence of myocardial or pericardial injury supports the evidence for a physiological substrate underpinning the observed ECG changes. The use of ECG for screening athletes before ultra-endurance competition may aid in the identification of disease after prolonged exercise and avoid misdiagnosis and mismanagement of the athlete.

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REFERENCES

1 **O'Connor F**, Pyne S, Brennan F, *et al*. Exercise associated collapse: an algorithmic approach to race day management. *Am J Med Sports* 2003;**5**:212–7.
2 **Sallis R**. Collapse in the endurance athlete. *Sports Sci Exchange* 2004;**12**:1–5.
3 **Dawson E**, George KP, Shave R, *et al*. Does the heart fatigue subsequent to prolonged exercise in humans? *Sports Med* 2003;**33**:365–80.
4 **Douglas PS**, O'Toole ML, Hiller DB, *et al*. Cardiac fatigue after prolonged exercise. *Circulation* 1987;**76**:1206–13.
5 **Whyte GP**, George K, Sharma S, *et al*. Cardiac fatigue following prolonged endurance exercise of differing distances. *Med Sci Sports Exerc* 2000;**32**:1067–72.
6 **Shave RE**, Whyte GP, George K, *et al*. Prolonged exercise should be considered alongside typical symptoms of AMI when evaluating elevations in cardiac troponin T. *Heart* 2005;**91**:1219–20.
7 **Newby, NL**. Markers of cardiac ischemia, injury, and inflammation. *Prog Cardiovasc Dis* 2004;**46**:404–16.
8 **Sharma S**, Whyte G, Elliott PM, *et al*. Electrocardiographic changes in 1000 highly trained elite athletes. *Br J Sports Med* 1999;**30**:319–24.

COMMENTARY

Most cases of collapse after exercise are due to the persistence into recovery of a state of low peripheral resistance, to which ''dehydration'' probably plays no meaningful part. Thus the optimum treatment of collapse after exercise is to nurse the patient in the head-down position. This is usually remarkably effective, returning the systolic blood pressure to above 100 mm Hg and instantly reversing any symptoms of postural hypotension.

Had the physicians who first encountered this patient been aware that some athletes have a great capacity for peripheral vasodilatation and the development of postural hypotension under specific conditions, the patient would have been spared unnecessary interventions and the costs the authors describe. There is an old saying that athletes should only ever accept the opinion of a doctor who is also an athlete, or at least has some passing understanding of sports medicine.

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